A DISSERTATION ON THE COMPARATIVE STUDY OF THE EFFICACY OF VARIOUS TOPICAL TREATMENT MODALITIES IN PALMOPLANTAR PSORIASIS

Dissertation Submitted to

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(BRANCH XII A)

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CERTIFICATE

Certified that this dissertation entitled **"Comparative Study of the efficacy of various topical treatment modalities in Palmoplantar psoriasis"** is a bonafide work done by **DR. V.RENUKA, Post graduate student of the Department of Dermatology and Leprology and Institute of Venereology, Madras Medical College, Chennai- 3**, during the academic year 2004 – 2007. This work has not previously formed the basis for the award of any degree or diploma**.**

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Declaration

I, **Dr. V.RENUKA,** solemnly declare that dissertation titled, **"COMPARATIVE STUDY OF THE EFFICACY OF VARIOUS TOPICAL TREATMENT MODALITIES IN PALMOPLANTAR PSORIASIS"** is a bonafide work done by me at Madras Medical College during 2004-2007 under the guidance and supervision of **Prof. Dr. B. PARVEEN, M.D.,D.D.,** Professor and Head, Department of Dermatology, Madras Medical College, Chennai-600 003.

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CONTENTS

INTRODUCTION

Psoriasis is a common, genetically determined, inflammatory and proliferative disease of the skin. The most characteristic lesions consist of red, scaly, sharply demarcated, indurated plaques present particularly on the elbows, knees, lowerback, extensor surfaces and scalp.

The first recognisable description of psoriasis is attributed to Celsus (25BC-45AD) in his de re medica nearly 2000 years ago. The disease was described under the heading of impetigo from the Latin word impeto which means "to attack or rush on" Galen was the first to use the word psoriasis from the Greek work 'psora' which means 'to itch'. Psoriasis and Leprosy were grouped together for centuries. Willan was the first to accurately describe psoriasis and its various manifestations in 1809, but he did not separate it with certainty from Leprosy. In 1841, Hebra definitively distinguished the clinical picture of psoriasis from that of Hansen's disease.

Eventhough a number of treatment modalities are available, psoriasis continues to be a therapeutic challenge in spite of our growing knowledge of its pathogenesis.

DIFFERENT CLINICAL TYPES OF PSORIASIS

Classical types	Psoriasis vulgaris
	Guttate psoriasis
	Pustular psoriasis
	Arthropathic psoriasis
	Erythrodermic psoriasis
Special types	Rupioid
	Ostraceous
	Elephantine
Atypical forms	Follicular
	Verrucous
	Lichenoid variety
	Linear
	Zonal
	Seborrhoeic
	Mucosal lesions
	Ocular lesions
According to site	Scalp
	Penis
	Flexural
	Nail
	Palms and Soles

Palmo plantar psoriasis is one of the type of psoriasis which can occur alone or along with the involvement of other areas.

In most cases the lesions are well defined but they are less scaly and the surface often shows fissures. It may be pustular or non pustular

Three forms of lesions can occur in palms and soles

1. Diffuse hyperkeratotic plaques
2. Erythematous patches or plaques studded with minute superficial pustules
3. Discrete scaly plaques or patches
4. Rarely Rupioid Lesions can occur on the soles with characteristic limpet like scales.

Palmo plantar psoriasis can also be classified as

1. Pustular - Acute pustular bacterid
   Chronic pustular bacterid
   Palmo plantar psoriasis with pustules
2. Non pustular - Diffuse
   Annular
   Delling or crateriform
   Marginal keratotic

In this study various topical modalities of treatment are used for palmoplantar psoriasis like

0.1% Betamethasone valerate ointment

0.05% Tazarotene gel

Topical PUVA using methoxypsoralen solution 1%

Short contact compound dithranol oint (dithranol 1.15%, salicyclic acid 1.15%, coal tar 5.3% in white soft paraffin)

Liquid paraffin.

There are numerous topical therapies available like coaltar, anthralin, methotrexate, vitamin D analogues tacrolimus, salicylic acid 2 - 10%. emollients, etc.

LITERATURE REVIEW

INTRODUCTION

Palmo plantar psoriasis has various therapeutic options both topical and systemic. The topical modalities of treatment generally used are

1. Coal tar
2. Dithranol
3. Topical corticosteroids
4. Vitamin D analogues
    a. Calcitriol
    b. Calcipotriol
    c. Tacalcitol
    d. Maxacalcitol
5. Topical psoralen
6. Topical retinoid (Tazarotene)
7. Topical cytostatic therapy
    a. Mechlorethamine (Nitrogen mustard)
    b. Thiotepa
    c. 5 - Flurouracil
    d. Lomustine
    e. Methotrexate

8. Tacrolimus (FK 506)

9. Emollients

10. Salicylic acid 2 - 10%

11. Arachidonic acid 0.5 - 2%

12. Topical allantoin

COAL TAR

Tar has been used in Topical therapy for more than a century

DIFFERENT TYPES OF TAR [1]

a. Shale tar

Icthammol

b. Wood tar

Juniper tar

Pine tar

Pix liquida

c. Coal Tar

Coal tar solution

Strong coal tar solution

Coal tar is a complex mixture of thousands of substances produced by primary condensation during the carbonization of coal[2]. Some 400 known substances comprise 55% of Tar by weight.

PREPARATIONS

Coal tar is available as ointment, liquid, alcohol extract, gel, shampoo and soap. Each has its own used in specific areas. It is also available in combination with salicylic acid.

MECHANISM OF ACTION

1. Anti-Mitotic

Coal Tar has been found to depress mitosis and DNA synthesis by the production of DNA adducts and oxidation induced DNA damage in human mammary epithelial cells [3].

2. Phototoxicity

Goeckerman in 1925, described the enhancement of the therapeutic effect of tar after UV light exposure. The following components of coal tar act as photosensitizers, anthracene, fluoranthrene, phenanthrene, benzpyrene and benzacridine [4].

Ultraviolet fluorescence microscopy has proved that the hair follicles and the sebaceous glands may be an important route of penetration of the coal tar into the skin.

3. Sebostatic effect

It has been found that a 10% coal tar distillate reduces the size of the sebaceous glands and the number of mitoses. It is possible that this will lead to a subsequent reduction of the secretion of sebum [4].

4. Antifungal effect[5]

It has been demonstrated that coal tar has an antifungal potential on Malassezia furfur in vitro. This could, in part, be responsible for the response of seborrhoeic dermatitis and pityriasis capitis to coal tar [5].

INDICATIONS

Apart from its use in psoriasis, coal tar can also be used in pityriasis capitis and seborrhoeic dermatitis [4]. It has also been used in eczemas, especially atopic dermatitis [6].

ADVERSE EFFECTS

Irritation folliculitis and tar acne are the most frequent adverse effects of tar treatment. Phototoxic reactions are also common. Allergic contact dermatitis does occur but is rare.

Kennaway identified two carcinogens in coal tar, 3,4 benzpyrene and 1, 2-benzpyrene. A 2.4 fold increased risk of developing skin carcinomas was found in patients with high rate of exposure to tar and UV light [7].

PREPARATIONS

Coal tar is available in the form of ointments, creams, gels, scalp shampoos, solutions, lotions and suspensions.

COMBINATION THERAPIES

Coal tar can be combined with various other drugs for better efficacy.

1. Goeckermans regimen [8]

It consists of daily application of 2 - 5% crude tar, combined with a tar bath and UV light. Many modifications of the Goeckermans

regimen have been advocated using alcoholic extracts of tar in cream or ointment bases, tar gels, etc. which are easier to handle.

2. Coal tar and Dithranol [9]

A combination of 5% crude coal tar and dithranol was found to be as effective as dithranol alone when used in short contact treatment of psoriasis and was also less irritant.

3. Coal tar and topical steroids [10]

This combination reduces the irritation caused by coal tar.

4. Coal tar and salicylic acid.

Used in treatment of psoriasis

PRECAUTIONS

1. Coal tar application should be avoided over face, genitalia and flexures.

2. Coal tar therapy is contraindicated in erythrodermic and generalised pustular psoriasis.

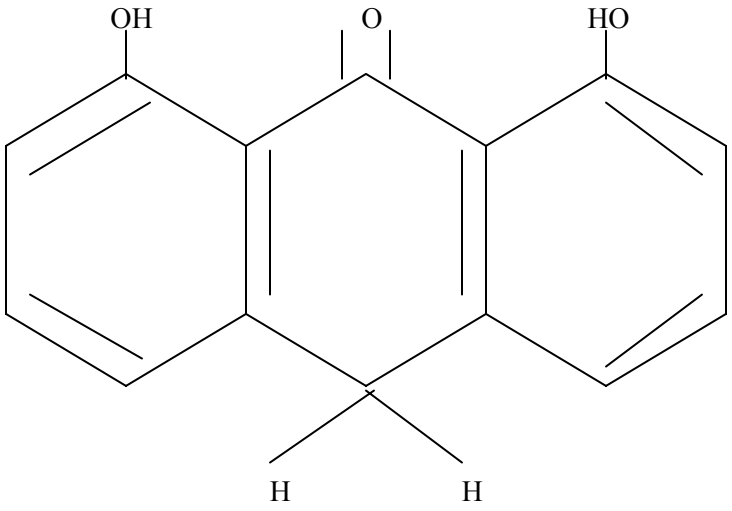
3. Pre existing folliculities or severe acne are also possible contraindications.

DITHRANOL

In history of medicine there are a few accidents proved to be good for mankind. One such is that of dithranol. Chrysarobin, 3-methyl dithranol,

obtained from araroba tree. The word araroba meaning ‘tawny coloured’. This is the active ingredient of Goa powder, imported by Portuguese from Brazil to India [11]. Following misdiagnosis it was accidentally used in psoriasis and found to be effective . It was called "Cignolin" in Germany and "Anthraline” in North America.

Structural Chemistry [13]



Anthralin has an unstable chemistry with 2 focal points: phenolic hydroxyl groups at carbon atoms 1 and 8 and two reactive hydrogen atoms on carbon ten. It undergoes auto-oxidation, which is both pH and light dependent.

MECHANISM OF ACTION

1. Antiproliferative effect

It induces a decrease in epidermal growth factor binding in a dose dependent manner. This may contribute to the anti psoriatic action of dithranol [14]

Dithranol also causes a marked decrease in transforming growth factor α MRNA expression on cultured human keratinocytes [15].

2. Action of anthralin on mitochondria

It interacts with the electron transport chain on the inner mitochondrial membrane resulting in a reduction of ATP Synthesis [13]

This loss of energy supply in keratinocytes including reduction of glucose 6 phosphate dehydrogenase could explain, at least in part, the therapeutic efficiency of anthralin in psoriasis.

3. Inhibition of biosynthesis of polyamine by anthralin

Ornithine decarboxylase catalyses the production of putrescine from ornithine and is associated with cellular proliferation. In psoriatic epidermis, polyamine levels are abnormally elevated, so anthralin inhibits this polyamine in psoriatic skin [13].

4.Effect of anthralin on cyclic nucleotides

Anthralin has also been shown to reduce the elevated levels of cyclic guanosine mono-phosphate (cGMP) that were present in involved skin. Although the precise mechanism is not known, there is much evidence to suggest that it acts on various cell regulatory systems [13].

5. Anti chemotactic activity of anthralin.

It is a potent inhibitor of leukotriene production and LTB4 - omega oxidation by human neutrophils thereby reducing the LTB4 - induced intra epidermal accumulation of polymorphonuclear leucocytes [16].

Also significantly lowers IFN - gamma and TNF alpha levels in psoriatic patients [17].

Anthralin has been found to cause significant fall in epidermal calmodulin levels in the psoriatic lesions associated with clearance of psoriatic lesions [18].

Anthralin also down regulates 12(S) hydroxy ecosotetraenoic acid (12 (S) HETE) receptors on epidermal cells which may contribute to its antipsoriatic action [19].

INDICATIONS

1. Psoriasis - both as monotherapy and in combination with various other therapies. It can also be used in nail and childhood psoriasis.

2. Facial seborrhoeic dermatitis in low-dose preparations [20]. At higher concentrations shown to inhibit growth of pityrosporum ovale [21].

3. Inflammatory linear verrucous epidermal naevus [22].

4. 2% use in verruca vulgaris [23]

5. Alopecia areata [24]

PREPARATIONS

Anthralin is available as paste, ointment, cream and sticks.

MODIFICATIONS OF ANTHRALIN THERAPY

1. Short contact therapy [11,12]

With the hope of avoiding the disadvantages, higher strength of dithranol is kept in contact with lesion for a short period. Concentrations ranging from 0.5 to 3% and even upto 8% have been tried as short contact therapy for 10 -20 minutes and have been found to be successful [25].

2. Ingram regimen

First the patient soaks and washes thoroughly in 90 litre (20 gallon) bath to which 120 ml of alcoholic solution of coal tar and 30ml of teepol is added. After drying, small doses of UVB are given, increasing gradually to suberythmogenic dose. Then the lesion is accurately covered by application of dithranol 0.42% in stiffened

lassars paste, which is powdered with talc and covered with stockinet dressing. The patient then dresses and pursues his work, returning for treatment after 24 hours. After 24 hours, the paste being removed with olive oil prior to bathing the following day [13].

Disadvantages of this treatment

1. Requires hospitalization
2. Staining of the lesion and dress
3. Irritation at the site

3. Liposomal delivery of dithranol.

There were no reports of lesional or perilesional irritation and only one patient showed faint brown staining of the skin [26].

4. Micanol

In this dithranol is micro encapsulated in crystalline monoglyceride.

This preparation is easy to wash off and staining and irritation are inconspicuocs. Hence, it can be used in out patient setting [27].

COMBINATION THERAPIES

Anthralin can be used in combination with

a. PUVA

b. Narrowband UVB

c. Coal tar

d. Topical steroids

e. Calcipotriol

f. Tazarotene

g. Oral cyclosporine

h. Oral retinoids

ADVERSE EFFECTS

1. Anthralin erythema is due to release of prostaglandin and superoxide radicals released from C1O methylene group of anthralin and anthralin radicals produced in the skin [28].

2. Staining and pigmentation is due to formation of anthraquinone oxidation products.

3. Rarely skin cancer

4. Ocular damage

5. Allergic contact dermatitis

PRECAUTIONS

1. Should be applied only to psoriatic plaques and not to normal skin

2. Better avoided in face.

3. Contra-indicated in acute, unstable, generalised pustular and erythodermic psoriasis.

CONCLUSION

Anthralin has been available for many years and has been an useful anti-psoriatic agent. It must be used with care and instructions for its use are required for both physicians and patients. The introduction of short contact therapies may make this compound more useful in treatment of palmoplantar psoriasis.

TAZAROTENE

Tazarotene is a third generation receptor selective polyaromatic, synthetic retinoid also known as Arotenoid. It is a prodrug of a water soluble active metabolite called tazarotenic acid. The conversion takes place in the skin by the help of an enzyme called esterase.

It has high affinity for RAR β γ than for RAR α. But no affinity for RXR.

MECHANISM OF ACTION

Down regulates abnormal expression of keratinocyte transglutaminase (T gase 1), epidermal growth factor receptor and hyper proliferative keratin K6/K16.

Blocks the induction of ornithine decarboxylase activity, which is associated with cell proliferation and hyperplasia.

Inhibits cornified as well as cross linked envelope formation.

Decreases migration inhibitory factor related protein (MRP-8) a marker of inflammation in psoriasis[29].

Up regulates tazarotene inducible genes (TG 1, 2, 3) and has antiproliferative properties

PHARMACO KINETICS

Systemic absorbtion is negligible because of rapid metabolism (less than 20 minutes) to hydrophilic metabolite and limited percutaneous penetration. Onset of action is within two weeks and elimination half life is 17 to 18 hrs.

Excreted through skin, urine and faeces. It is available as cream or gel (0.05% and 0.1%)

PRECAUTIONS

Simultaneous use of tazarotene should be avoided with abrasive or medicated soap, products containing high concentration of alcohol, astringents, medications or cosmetics having strong drying effects

INDICATIONS

Mild to moderate psorasis involving 10% - 20% of body surface area[30]

Acne vulgaris

Photoaging (Wrinkling, mottled pigmentation, facial roughness)

Cutaneous T cell lymphoma

Disorders of keratinization (Dariers disease, pityriasis rubra pilaris, ichthyosis)

Treat and prevent skin cancer (Basal cell carcinoma, Xeroderma pigmentosum) [31]

CONTRAINDICATION

Pregnancy

Lactation

SIDE EFFECTS

Skin irritation, which is characterized by erythema, peeling, desquamation, dryness, burning and pruritus[32]

Hypo or hyper pigmentation

Koebnerization of psoriasis

TOPICAL CORTICOSTEROIDS

INTRODUCTION

The revolution brought about in dermatological therapy by the introduction of topical corticosteroids, which started with compound F or hydrocortisone in 1952, is well known. Topical steroids play an important role in the treatment of psorasis particularly when combined with other modalities of therapy.

STRUCTURE

Hydrocortisone is the parent compound of modern glucocortico steroid derivatives. It has a cyclopentanoperhydrophenanthrene nucleus, which has been chemically modified over the years to enhance glucocorticosteroid activity and minimise mineralocorticoid effects[33].

Factors affecting the potency of topical corticosteroids are lipophilicity of the molecule, additional double bond at 1, 2 position, addition of halide at 9, 6, or 21 position and the state of the epidermal barrier. Halogenation of the 9 position will significantly increase glucocorticoid activity.

CLASSIFICATION[34]

The introduction of the vasoconstrictor assay has proved of great value in the classification of topical corticosteroids. The assay depends upon the property of corticosteroids to produce transient vasoconstriction. Steriod potency can also be assessed by Duhring chamber.

The corticosteroids are classified into the following groups based on their potency.

1. Very potent	: Clobetasol propionate 0.05%
	Halcinonide 0.5%
	Beclomethasone dipropionate 0.05%
2. Potent	: Beclomethasone dipropionate 0.025%
	Betamethasone valerate 0.1%
	Fluocinolone acetonide 0.05%
	Fluocinonide 0.05%
	Fluticasone propionate 0.05%
	Momentasone furoate 0.1%
3. Moderately Potent	: Triamcinolone acetonide 0.1%
	Hydrocortisone butyrate 0.1%
	Desonide 0.5%
	Flurandrenolone 0.05%
4. Mildly Potent	: Clobetasole butyrate 0.05%
	Fluocortolone hexonate 0.1%
5. Weak	: Hydrocortisone 0.1 - 2.5%
	Methyl Prednisolone 0.025%

MECHANISM OF ACTION

Corticosteroids have anti-inflammatory, immunosuppressive and antimitogenic activities due to their ability to exert multiple effects on the various functions of leucocytes, epidermal and dermal cells[35].

Corticosteroids diffuse through the stratum corneum barrier and through the cell membranes reach the cytoplasm. In the cytoplasm they bind with glucocorticoid receptor α (GRα) and then enters the nuclear compartment and interacts with glucocorticoid responsive elements on the genome.

In addition, the ligand bound receptor can inhibit directly or indirectly, the activity of other transcription factors like NFKB, AP-1 and NFAT[36].

These interactions leads to suppression of the production of inflammatory cytokines, inhibition of T cell activation, changes in the function of endothelial cells, granulocytes, mastcells, fibroblast and inhibition of proliferation.

They induce synthesis of lipocortin that regulate the activity of phospholipase A2. This enzyme affects the production of arachidonic acid, the precursor for leukotrienes and prostaglandins[37].

There is one more receptor in the cytoplasm called the GRβ, which is an endogenous inhibitor of glucocorticoid action[38]. Staphylococcal superantigen can upregulate expression of GRβ, providing a potential mechanism by which these bacteria might induce corticosteroid resistance[39].

THERAPEUTIC USES

Papulosquamous disorder.

1. Psoriasis
2. Lichen planus[40]
3. Reiters disease

Eczemas

1. Atopic dermatitis[41]
2. Seborrhoeic dermatitis[42]
3. Contact dermatitis (allergic, irritant) [43]

4. Photocontact dermatitis
5. Lichen simplex chronicus
6. Actinic reticuloid[43]
7. Pompholyx[42]
8. Asteatotic eczema

Bullous disorders

1. Pemphigus group[43]
2. Bullous pemphigoid[46]
3. Epidermolysis bullosa
4. Sub corneal pustular dermatosis

Collagen vascular diseases

1. Lupus erythematosus
2. Dermatomyositis

Miscellaneous

1. Alopecia areata
2. Hemangiomas
3. Mycosis fungoides[44]
4. Sarcoidosis

5. Vitiligo

6. Cutaneous amyloidosis

7. Keloid / Hypertrophic scar

8. Aphthous stomatitis

9. Mastocytosis and Urticaria pigmentosa

10. Acne rosacea

11. Vasculitis, etc.

PREPARATIONS

They are available as ointments, creams, lotions, gels, foams, sprays and tapes[46].

COMBINATION THERAPIES

Topical corticosteroids have been used in combination with various other topical and systemic agents in the treatment of psoriasis. They are

1. Coal tar[47]

2. Anthralin

3. Calcipotriol[48]

4. Tazarotene[49]

5. PUVA[50]

ADVERSE EFFECTS

Epidermal	:	Thinning of epidermis
		Reduction in the size of epidermal cells
		Reduction in the metabolic activity
		Reduction in the number of cell layers
		Inhibition of melanocyte function with hypopigmentation
Dermal	:	Resorption of mucopolysaccharide ground substance with atrophy[51]
		Fragility of skin
		Suppression of collagen synthesis, so striae
Vasculature	:	Loss of connective tissue support leading to
		Erythema
		Telangiectasia[52]
		Purpura

Miscellaneous

Perioral dermatitis[52]

Acneiform eruption

Acne rosacea

Glaucoma

Cataracts and retinal detachment[53]

Allergic contact dermatitis[54]

Hypertrichosis

Infantile gluteal granulomas

Delayed wound healing

Cutaneous lymphangiectases[55]

Tachyphylaxis.

SYSTEMIC

Suppression of the pitutary - adrenal axis can occur with virtually any topical steroid especially potent steroids, leading to cushing syndrome[56]

PRECAUTIONS

No more than 45 gm per week of moderate to potent steroids in adults and 15gm per week in young children and infants are recommended for topical use.

Corticosteroids on continued use exhibit tachyphylaxis, the development of acute tolerance.

Sudden withdrawal of topical steroids in psoriasis can precipitate pustular psoriasis.

Topical steroids are to be used with caution over the face.

CONCLUSION

Topical steroids play an important role in the treatment of psoriasis. They should be used judiciously because of the possibility of rebound phenomenon when they are discontinued as well as the potential development of topical and systemic adverse effects. They can play an important role as an adjunct to other forms of therapy in the treatment of psoriasis.

SALICYLIC ACID

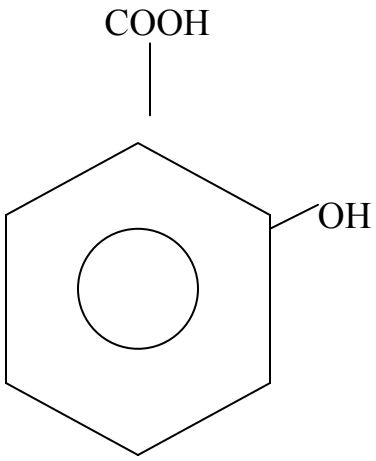
INTRODUCTION

Willow bark (Salix alba) is an ancient herbal remedy. The chemical structure of its active ingredient, salicylic acid (2-hydroxy-benzoic acid) was identified in 1838 [57]

Salicylic acid is a crystalline powder. It is lipid soluble and hence miscible with epidermal lipids and sebaceous gland lipids.

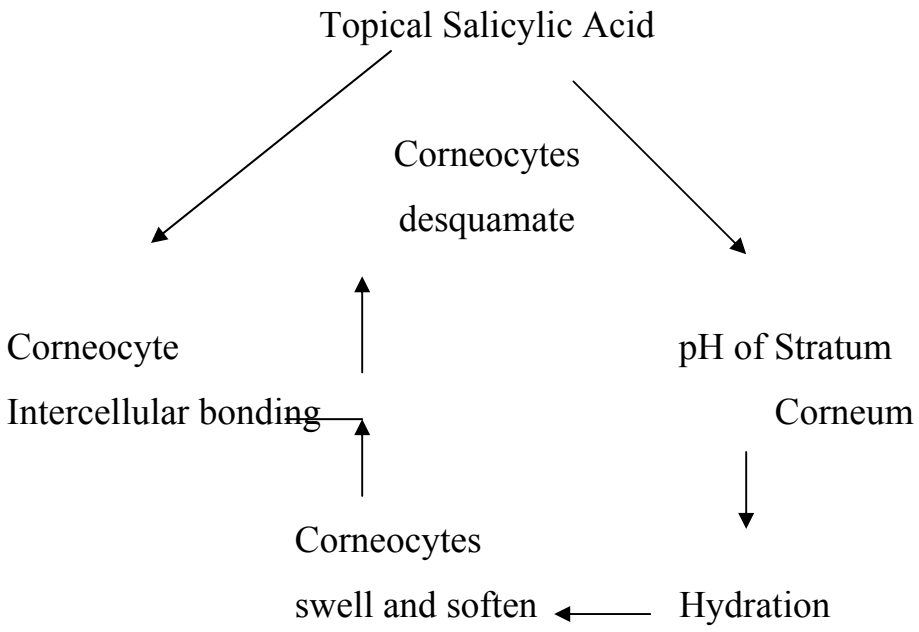
To obtain significant keratolytic effect salicylic acid must be formulated at a proper pH so that there will be enough of free acid.

STRUCTURE



MECHANISM OF ACTION

Reduction of corneocyte adhesion, perhaps by an action on the cement substance. This results in enhanced shedding of corneocytes and has no effect on mitotic activity[58].



Salicylic acid thus can cause enhancement of penetration of other chemicals like topical corticosteroids.

Salicylic acid can be photoprotective[59].

It also has bacteriostatic and bactericidal activity against yeast, gram negative and gram positive bacteria[57]

INDICATIONS

Psoriasis[60]

Seborrhoeic dermatitis[58]

Ichthiosis[58]

Warts[61]

Corns and calluses[58]

Acne[58]

Pityriasis rubra pilaris[62]

Keratodermas[58]

Dermatophyte infections[58]

Avulsion of toe nails[58]

Sunscreens[63]

Alopecia[64]

PREPARATIONS

Salicylic acid is available in

Collodion - based paints and gels

Shampoos

Ointments

Paste (Lassars paste contains salicylic acid and zinc oxide)

Creams

COMBINATION THERAPIES

Salicylic acid is used commonly in combination with various other topical therapeutic agents like,

Corticosteroids[65]

Coal tar

Other Keratolytics — Lactic acid

Propylene glycol

Urea[66]

Sulphur

Benzoic acid (Whitfield ointment)

Zinc oxide (Lassars paste)

Anthralin (Salicylic acid prevents the oxidation of anthralin)

Calcipotriol[67]

ADVERSE EFFECTS

Irritation and maceration at high concentrations

Contact sensitization

Systemic toxicity (Salicylism) [68]

The manifestations include nausea, vomiting, confusion, dizziness, delerium, psychosis, stupor, coma and death. Can also cause marked hyper ventilation and respiratory alkalosis.

PRECAUTIONS

Increases the toxicity of other topical agents because of enhanced absorption and hence should be used with caution.

Prolonged use should be avoided to prevent salicylism.

Used with caution in patients with hepatic and renal impairment especially young children.

It blocks UVB and inactivates calcipotriol if used concurrently[69].

CONCLUSION

Salicylic acid, especially when used cautiously, is an effective and safe antipsoriatic agent.

TOPICAL PUVA THERAPY

Psoralen photochemotherapy is a combination of psoralen (P) and long wave UVR (PUVA) that brings about a therapeutically beneficial result not produced by either the drug or radiation alone [70,71].

Application of 8-MOP in creams, ointments or lotions followed by UVR is effective in clearing psoriasis but has several disadvantages.

PSORALEN PHOTO CHEMISTRY

Ground state psoralen molecules are activated to the excited singlet state by absorption of photons in the UVA wave band. The peak of the absorption spectrum is around 320 – 330 nm. Two types of photo chemical reaction occur[72].

Type 1 – (Direct) resulting in mono functional adducts in DNA strands.

Type-2 – (Indirect) reactions resulting in production of reactive O_2 species and free-radicals that cause damage to cell membrane and cytoplasmic constituents[73].

MECHANISM OF ACTION

Suppersion of DNA Synthesis, through formation of mono and bi functional adducts [74], which occurs immediately. Subsequently cell proliferation is also inhibited in patients with psoriasis who receive repeated PUVA treatment.

IMMUNOLOGIC EFFECTS

PUVA down regulate certain lymphocytes and antigen presenting cells and influences adhesion molecule expression [75].

Selective cytotoxicity through production of free radicals

Stimulation of melanocyte proliferation and increased tyrosinase synthesis.

INDICATIONS [76-79]

Psoriasis[80,81]

Vitiligo

Mycosis fungoides

Lymphomatoid papulosis

Pityriasis lichenoides

Langerhans cell histiocytosis

Atopic dermatitis[82]

Seborrhoeic dermatitis

Pityriasis rubra pilaris

Lichen nitidus

Alopecia areata

Urticaria pigmentosa

Actinic prurigo[83]

MODE OF ADMINISTRATION

Applied in the form of lotion and creams. A lotion containing 0.1 to 1.0 % 8 methoxypsoralen is used. Then the area should be exposed to UVA. This treatment is given twice weekly with UVA dose being gradually increased.

The hands should be immediately washed and topical emollients should be applied and further photo protection from sunlight insisted using sun screeners.

LIGHT SOURCES FOR UVA

UVA radiation spectrum ranges from 320 to 400 nm. The PUVA action spectrum is defined as the effectiveness of clearing psoralen sensitized psoriasis as a function of wave length. Currently broad band high intensity UVA sources ranging from 320 to 400 nm with a peak emission at 352 nm are typically used in PUVA.

However recent advances demonstrate the peak action spectrum of psoriasis to be around 330 nm.

The commonly used lamps are [84]

1. Fluorescent lamps
2. Metal halide lamps

PHOTO SENSITIVE EFFECTS OF PUVA

PUVA produces an inflammatory response that manifest as

1. Delayed phototoxic erythema [85,86]
2. Intense pruritus
3. Pigmentation

These reactions are related to the dose of the drug and of UVA and to the individual sensitivity to phototoxic reaction.

TREATMENT PROCEDURE

Before starting a patient on PUVA therapy contra indication to it have to be excluded.

CONTRA INDICATION [87]

ABSOLUTE

1. Xeroderma pigmentosum, Blooms syndrome and other congenital photo sensitivity disorders
2. Lupus erythematosus
3. Pemphigus, pemphigoid [88]
4. History of idiosyncratic reaction to psoralen compounds

RELATIVE

1. Concurrent photo sensitizing medication
2. Prior exposure to ionizing radiation or arsenic[89]
3. History of skin cancer/chronic actinic damage[89]
4. History or family history or melanomas
5. Pregnancy, lactation [90]
6. Age less than 18 years
7. Significant renal, hepatic or cardiac disfunction
8. Cataract

ADVERSE EFFECT[77]

SHORT TERM

A. Photo toxic reactions

   1. Erythema
   2. Pruritus
   3. Pain
   4. Blistering on hands and feet
   5. Photonycholysis
   6. Phytophoto dermatitis

B. Due to psoralen

1. Nausea, vomiting
2. Head ache
3. Dizziness
4. Broncho constriction
5. Hepato toxictity
6. Drug fever
7. Exanthems

C. Unsual side effect

1. Hypertrichosis of face
2. Subungual haemorrhage of finger nails
3. Acneiform eruptions
4. Induction of bullous pemphigoid

LONG TERM ADVERSE EFFECT

1. Chronic actinic damage
2. Carcinogenesis – Squamous cell carcinoma [91,92]

   - Melanomas
3. Cataract
4. Immunological

Advantages

No systemic side effects such as nausea and no carcinogenic effects.

Disadvantages

1. Topical PUVA is laborious and time consuming if every lesion has to be treated individually
2. The formation of erythema and blisters is more common with topical psoralen application
3. Intense irregular pigmentation may be seen at the site of treated plaques

AIM OF THE STUDY

BACKGROUND

Palmoplantar psoriasis is a chronic disease with remissions and exacerbations. Most of the topical therapies currently available for psoriasis are either suited for short term therapy or long term maintenance therapy. Furthermore topical corticosteroids commonly used for palmplantar psoriasis, show diminished response on continuous use due to tachypylaxis and more incidence of recurrence.

OBJECTIVE

To compare the efficacy of various topical therapies like

Short contact compound dithranol ointment (dithranol 1.15%, salicylic acid 1.15%, coal tar solution 5.3% in white soft paraffin)

Topical 0.1% Betamethasone valerate ointment

Topical tazarotene 0.05% gel

Topical PUVA using -1% methoxypsoralen solution

Liquid paraffin.

MATERIALS AND METHODS

100 patients who attended the psoriasis out patient clinic at the department of Dermatology, Government General Hospital, Chennai from September 2004 to September 2006 were included in the study. The diagnosis was made on clinical grounds and histopathological examination.

INCLUSION CRITERIA

Patients with stable palmoplantar psoriasis

Patients who has not used other forms of topical therapy during the previous 4 weeks.

For topical PUVA therapy age more than 18years was considered.

EXCLUSION CRITERIA

Palmoplantar psoriasis along with other body surface area involvement.

Pregnancy and lactation

Unwillingness to give consent for return for weekly evaluation.

Patients who have had other forms of therapy during the previous 4 weeks.

PATIENT EVALUATION

History

General Examination

Systemic Examination

Dermatological Examination

Laboratory Investigations

a. Baseline Haemogram

b. Urinary Analysis

c. Biopsy in doubtful cases

TREATMENT PROTOCOL

100 patients were randomly allocated to 5 groups (20 patients each) to receive topical 0.1% betamethasone valerate, 0.05% tazarotene, short contact compound dithranol, topical PUVA and liquid paraffin respectively.

Topical 0.1% betamethasone valerate was applied twice daily.

0.05% tazarotene was applied once a day at bed time.

Short contact compound dithranol was used for 20 minutes once a day.

Topical PUVA was given thrice weekly after applying 1% methoxypsoralen.

Liquid paraffin was used three times daily.

TREATMENT EVALUATION

Severity and extent of psoriasis were evaluated at pretreatment baseline (Day 0) and then at weekly intervals for 4 weeks, followed fortnightly for 12 weeks, followed by monthly intervals up to 24 weeks by means of "Psoriasis Area and Severity Index" (PASI).

Severity of erythema (E), Desquamation (D) and Induration (I) was recorded on a 5-point scale as follows

0 - Nil
1 - Mild
2 - Moderate
3 - Severe
4 - Very severe

Area of involvement for palms and soles was taken as 1 i.e less than 10% involvement

PASI was calculated as

$$PASI = 0.2 (EU + IU + DU) AU + 0.4 (EL + IL + DL) AL$$

U - Palms (Upperlimb)

L - Soles (Lower limb)

At each weekly visit, the patients were asked about complaints and adverse effects like itching or burning sensation in lesional or perilesional skin. Patients were also examined for perilesional erythema.

OBSERVATIONS AND RESULTS

AGE DISTRIBUTION

The mean age in this study was 43.35. The range was from 10 to 85 years. The age distribution is depicted in Fig.1

SEX DISTRIBUTION

In patients chosen for steroids and dithranol, the males and females were equal, where as for placebo and tazarotene males outnumbered females and for PUVA, females outnumbered males. (Fig. 2)

The overall male to female ratio was 52 :48. (Fig. 3)

DURATION OF ILLNESS

The mean duration of psoriasis among the patients was in the range between 39.11 and 25.65 months. (Fig. 4)

EXACERBATING FACTORS

There was no exacerbating factor for 86% of the study group. Among the most common exacerbating factor, stress, cold and menopause, 9% was for stress and 3% & 2% was for cold and menopause respectively. (Fig. 5)

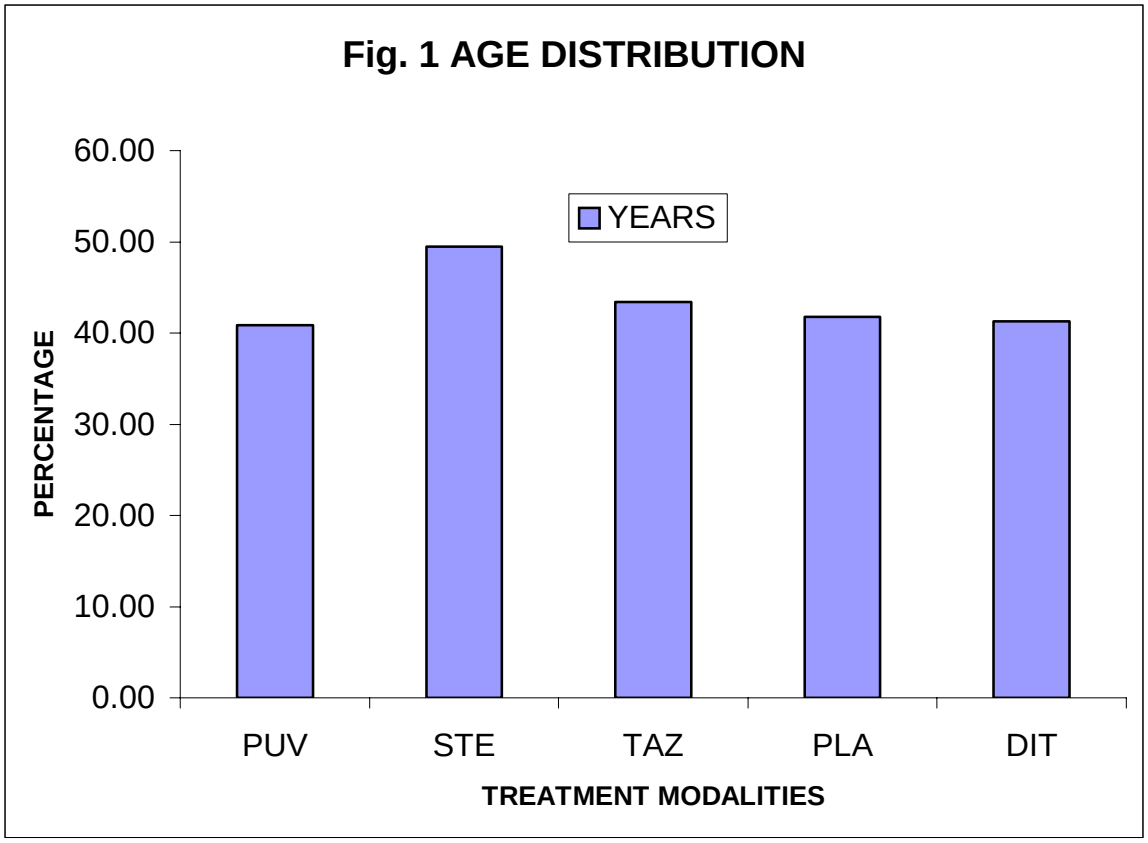


Fig. 1 AGE DISTRIBUTION
60.00
50.00
40.00
30.00
20.00
10.00
0.00
PERCENTAGE
YEARS
PUV
STE
TAZ
PLA
DIT
TREATMENT MODALITIES

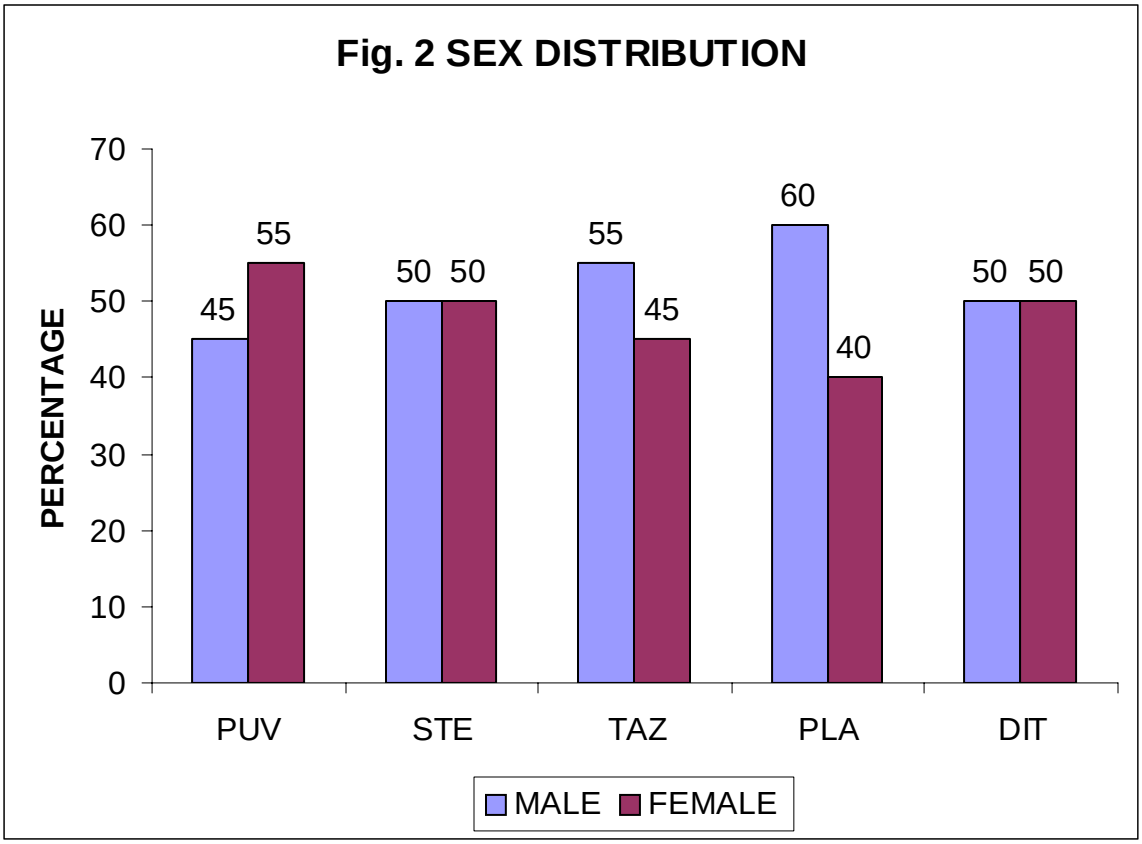


Fig. 2 SEX DISTRIBUTION
70
60
50
40
30
20
10
0
PERCENTAGE
45
55
50
50
55
45
60
40
50
50
PUV
STE
TAZ
PLA
DIT
MALE
FEMALE

Fig. 3 SEX RATIO

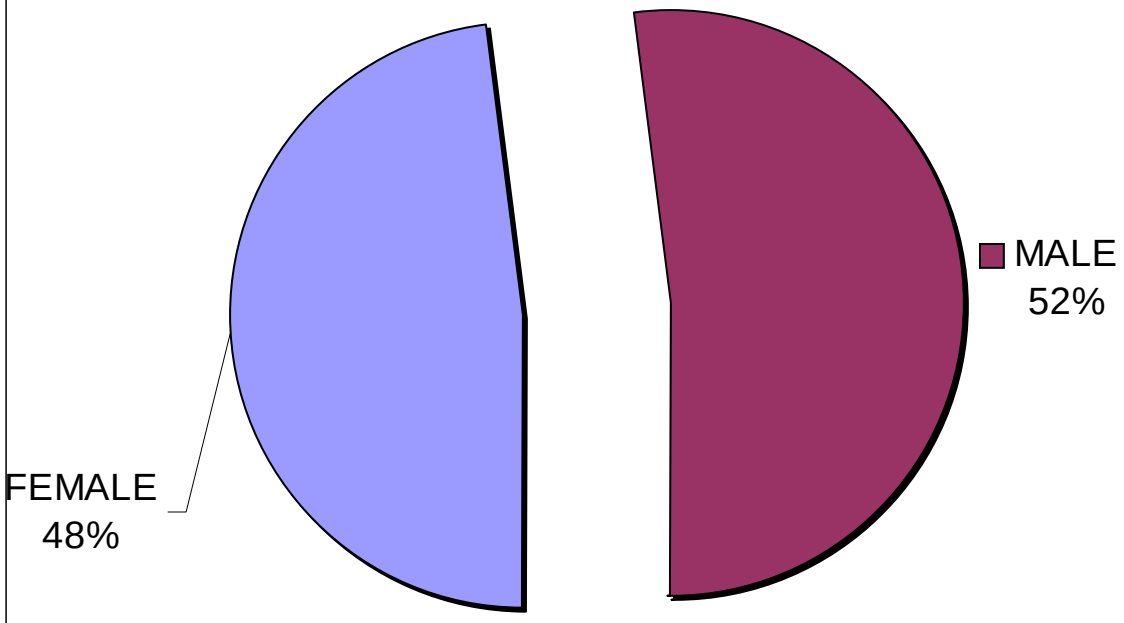
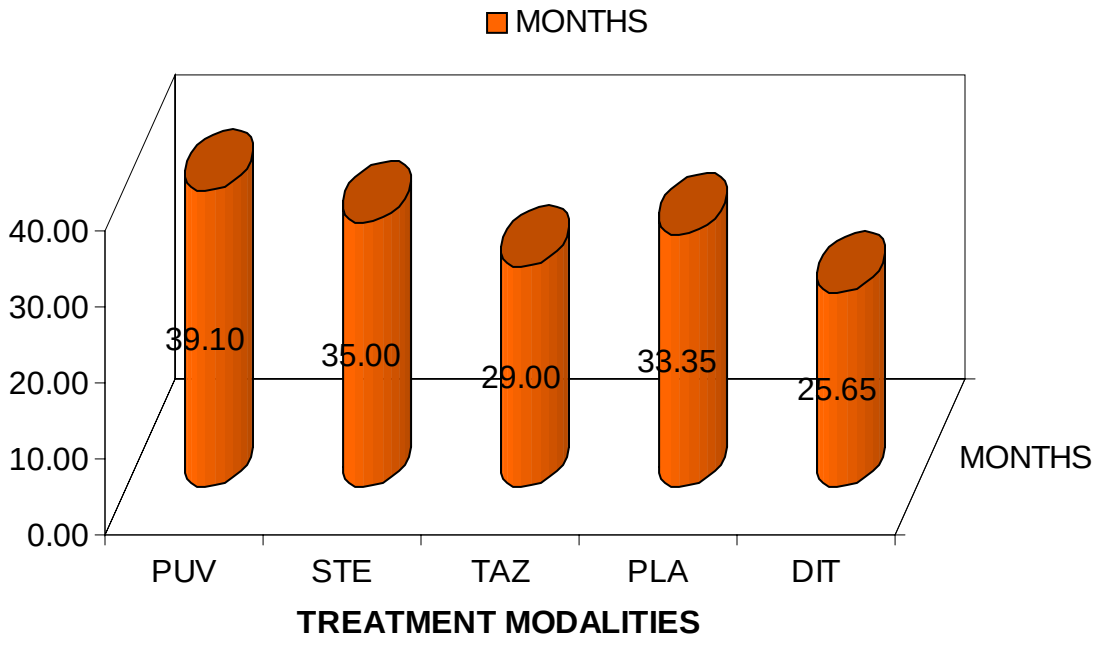


Fig. 4 DURATION OF PSORIASIS



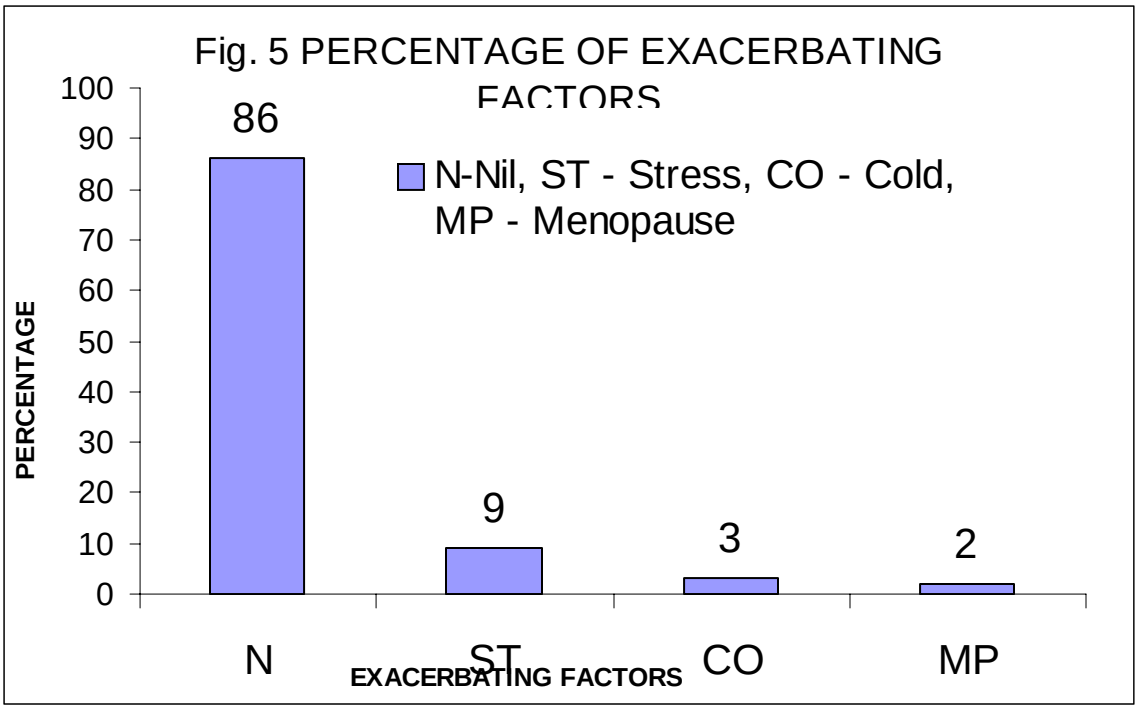


Fig. 5 PERCENTAGE OF EXACERBATING FACTORS
N-Nil, ST - Stress, CO - Cold, MP - Menopause
PERCENTAGE
100
90
80
70
60
50
40
30
20
10
0
86
9
3
2
N
ST
CO
MP
EXACERBATING FACTORS

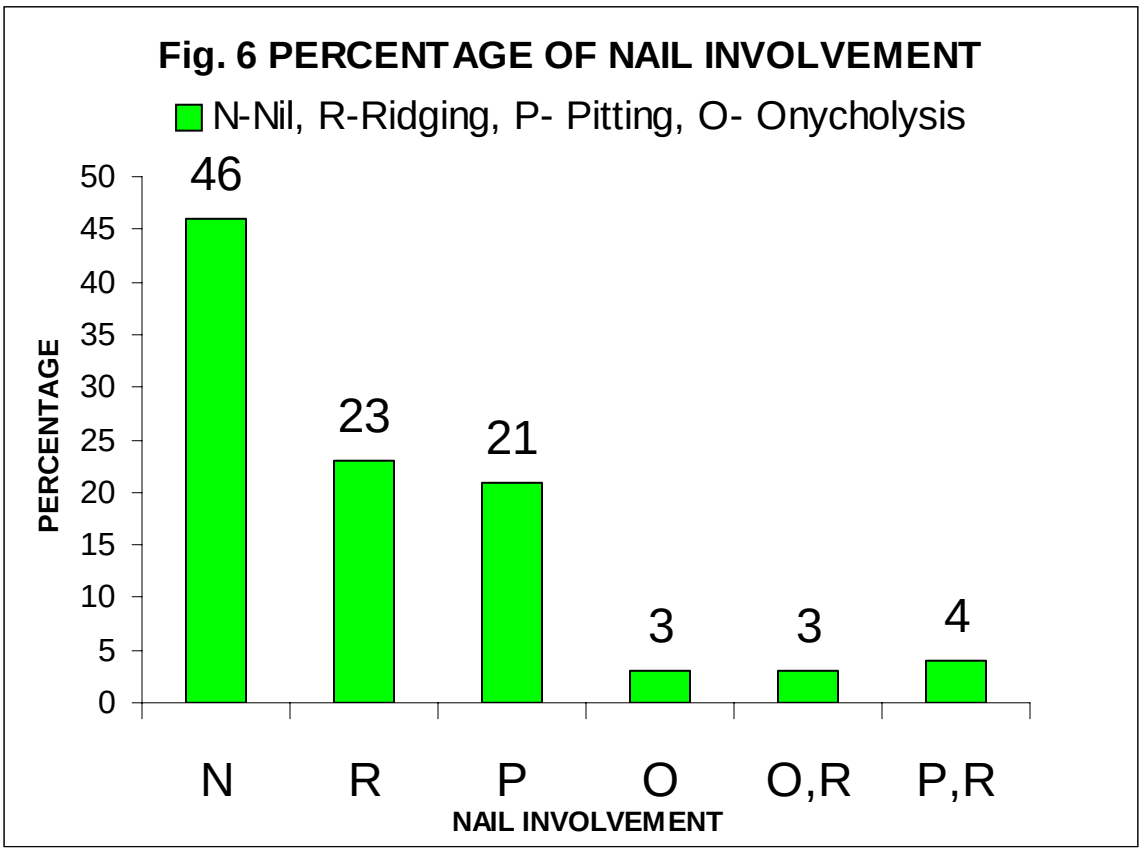


Fig. 6 PERCENTAGE OF NAIL INVOLVEMENT
N-Nil, R-Ridging, P- Pitting, O- Onycholysis
PERCENTAGE
50
45
40
35
30
25
20
15
10
5
0
46
23
21
3
3
4
N
R
P
O
O,R
P,R
NAIL INVOLVEMENT

FAMILY HISTORY

Family history of psoriasis was present among 3% of total patients, 1% each in placebo, tazarotene, dithranol groups and none in the PUVA and steroid groups.

NAIL CHANGES

46% had no nail changes. Among the others 23% had ridging, 21% had pitting, 3% had onycholysis, 4% pitting and ridging and 3% had onycholysis and ridging. (Fig. 6)

FOCAL SEPSIS

17% had evidence of focal sepsis in the ear, nose and throat and dental sepsis in the form of gingivitis, which was treated before the onset of therapy.

PASI REDUCTION

Fig. 7 depicts the reduction in the PASI scores obtained in the 5 groups at 0,4,8,12 and 24 weeks.

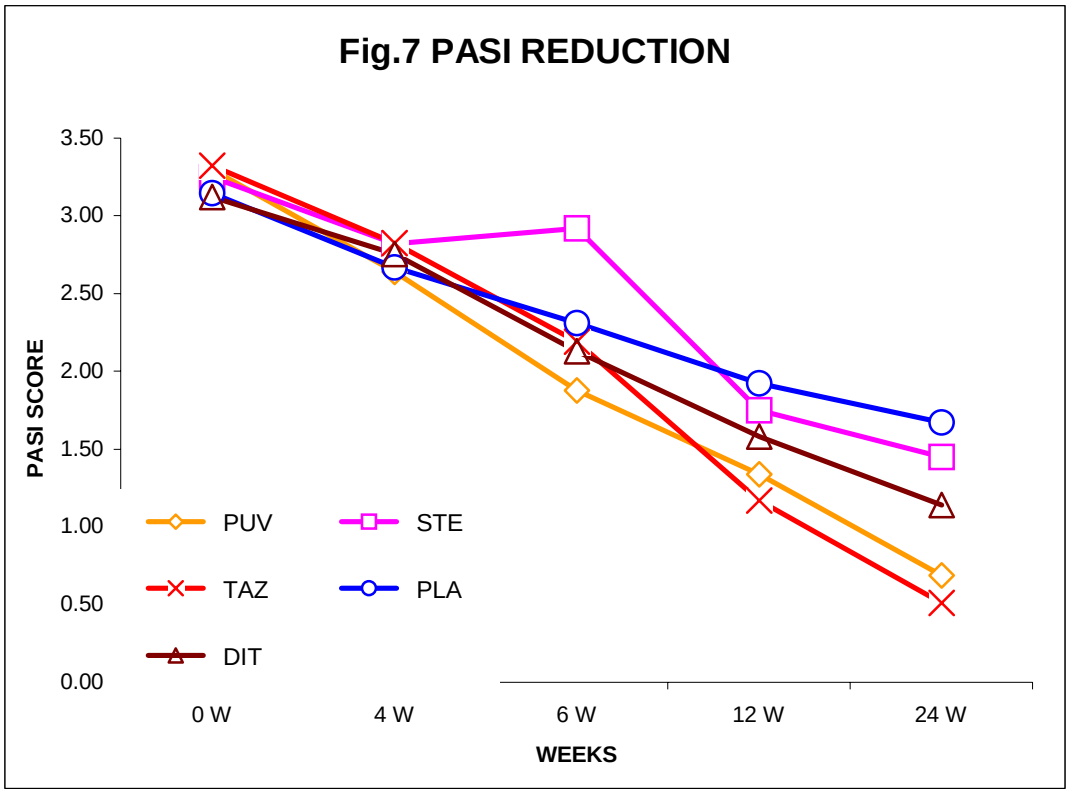


Fig.7 PASI REDUCTION
3.50
3.00
2.50
2.00
1.50
1.00
0.50
0.00
PASI SCORE
PUV
STE
TAZ
PLA
DIT
0 W
4 W
6 W
12 W
24 W
WEEKS

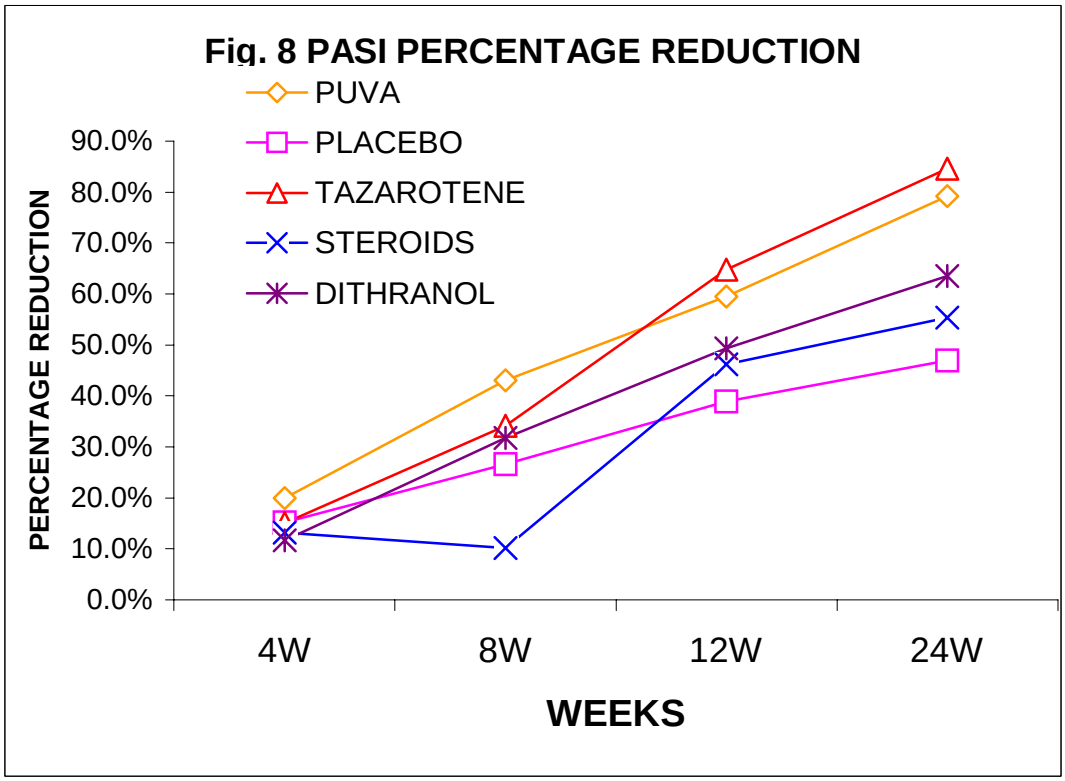


Fig. 8 PASI PERCENTAGE REDUCTION
PUVA
PLACEBO
TAZAROTENE
STEROIDS
DITHRANOL
90.0%
80.0%
70.0%
60.0%
50.0%
40.0%
30.0%
20.0%
10.0%
0.0%
PERCENTAGE REDUCTION
4W
8W
12W
24W
WEEKS

From the graph, the mean baseline PASI scores in the PUVA, steroid, tazarotene, placebo and dithranol were 3.3, 3.2, 3.3, 3.1 and 3.2 respectively. At 4 weeks of treatment, the mean PASI scores in 5 groups were 2.6, 2.8, 2.8, 2.6 and 2.7 respectively. At the end of 6^{th} and 12^{th} week there was substantial reduction in the mean PASI scores for all the groups. In the steroid group there was increase in PASI score between 4^{th} and 6^{th} week and thereafter reduction was observed up to the end of 24^{th} week. At the end of 24^{th} week the tazarotene and PUVA groups showed a sustained reduction in PASI score to 0.51 & 0.69 respectively. The other three groups showed a moderate reduction in PASI score between 1.14 and 1.67.

PERCENTAGE REDUCTION IN PASI

Fig. 8 depicts the percentage reduction in PASI scores in all the 5 groups at 4,8, 12 & 24^{th} week. At the end of 24^{th} week there was 84.66% reduction in PASI for tazarotene group followed by PUVA with 79.17%, dithranol with 63.46%, steroid with 55.38% and placebo with 46.94%.

ADVERSE EFFECTS

Adverse effects were found in all the groups except placebo. During 4^{th} to 6^{th} week of treatment, 25% of patients in steroid group showed exacerbation in the form of erythema. 15% of patients in tazarotene group

showed in the form of dryness and pruritus. 15% of patients of PUVA group showed in the form of erythema, polymorphic light eruption, burning sensation. 15% of patients in dithranol group showed in the form of burning sensation and pigmentation.

TOPICAL TAZAROTENE GROUP

Before therapy

The same patient after 12 weeks of treatment

TOPICAL TAZAROTENE GROUP

Before therapy

The same patient after 24 weeks of treatment

TOPICAL PUVA GROUP

Before therapy

The same patient after 12 weeks of treatment

TOPICAL PUVA GROUP

Before therapy

The same patient after 24 weeks of treatment

TOPICAL DITHRANOL GROUP

Before therapy

The same patient after 24 weeks of treatment

TOPICAL DITHRANOL GROUP

Before therapy

The same patient after 24 weeks of treatment

TOPICAL STEROID GROUP

Before therapy

The same patient after 24 weeks of treatment

TOPICAL STEROID GROUP

Before therapy

The same patient after 24 weeks of treatment

TOPICAL LIQUID PARAFFIN GROUP

Before therapy

The same patient after 12 weeks of treatment

ADVERSE EFFECTS

Erythema after PUVA therapy

Pigmentation after dithranol therapy

DISCUSSION

The study has shown that the mean age of the patients was 43.35 ranging from 10 to 85 years. The sex ratio was 52% male and 48% female. The various exacerbating factors in this study in descending order were stress, cold weather and menopause. Topical therapies like short contact compound dithranol, 0.1 % Betamethasone valerate, topical PUVA, 0.05 % tazarotene and liquid paraffin were used and reduction in PASI was assessed at every 4^{th} , 6^{th} , 12^{th} & 24^{th} weeks.

TAZAROTENE GROUP

Previous study on the topic "Management of topical modalities of psoriasis with special reference to Tazarotene by Dr.Susmit Haldar reported 50% of improvement by applying Tazarotene gel 0.05% twice daily at the end of 6weeks[30].

In this study, treatment made with the same 0.05% of Tazarotene gel once a day showed a maximum percentage reduction in PASI at the end of 8^{th}, 12^{th} and 24^{th} week was 34.14%, 64.81% and 84.66% respectively. Maximum improvement was observed between 12^{th} and 24^{th} week.

There was excellent compliance among the patients in this group and there were no defaulters. Patients also had minimal adverse effects like irritation, erythema, dryness and pruritus.

PUVA GROUP (TOPICAL)

In this study PUVA group showed the second best efficacy after tazarotene group showing a maximum reduction in PASI 79.17% at the end of 24 weeks as evidenced by flattening of plaques, decreased scaling and erythema[80,81]. This was consistent with the previous study conducted with topical application of methoxypsoralen plus UVA in which 67% of patients responded with considerable improvement.

15% of the patient had adverse effects in the form of polymorphic light eruption, erythema and burning sensation at the exposed areas. Except 4 defaulters, there was good compliance among the patients.

DITHRANOL GROUP

In this group, the percentage improvement in PASI was found to be 63.46% at the end of 24 weeks[12].

The study was consistent with the study conducted in UK by Gisslen H, Nordin P in which complete clearance of lesions was reported in 75% of the patients at the end of 24 weeks.

There was also good compliance in this group with no defaulters, however some adverse effects in the form of burning sensation and pigmentation were seen. However when the contact period was reduced to 10minutes the adverse effects reduced significantly

STEROID GROUP (0.1% BETAMETHASONE VALERATE GROUP)

Steroid group showed moderate efficacy with 55.38% improvement in PASI at the end of 24 weeks. However, there was no specific study reported using 0.1% betamethasonevalerate. There was good compliance among the patients with no defaulters. Few side effects like erythema and exacerbation were observed in some patients.

PLACEBO GROUP (LIQUID PARAFFIN)

In this group application of liquid paraffin showed 46.94% reduction of PASI at the end of 24weeks. There was good compliance among this group with no defaulters and no adverse effects.

The previous study has reported that use of liquid paraffin in palmo plantar psoriasis relieved feeling of dryness and pruritus[93]. There was no specific improvement or deterioration of the disease.

CONCLUSION

Topical therapies are the first line therapeutic strategy in the treatment of localized palmoplantar psoriasis and can be made effective when the appropriate drugs were used judiciously.

Among the five modalities compared in this study, tazarotene (0.05%) gel may be considered as an initial treatment of choice.

Topical PUVA is as effective as tazarotene except for the limiting factors for PUVA therapy such as availability of PUVA unit, patient compliance and long term side effects.

Topical dithranol is as effective as topical PUVA when used as 20minutes short contact therapy.

Topical 0.1% Betamethasone valerate was moderately effective with frequent exacerbation.

Liquid paraffin was the least effective with no adverse effects, no exacerbation and remissions. However it can be used as an adjunct with other topical therapies.

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COMPARATIVE STUDY OF THE EFFICACY OF VARIOUS TOPICAL TREATMENT MODALITIES IN PALMOPLANTAR PSORIASIS PROFORMA

Date

Name: Age: Sex:
Address:

Case No: Psoriasis clinic no:
Marital Status: Occupation:

History

Duration ☐ Years ☐ Months

Itching ☐ Yes ☐ No

Exacerbation with

☐ Cold Climate ☐ Sunlight ☐ Dialysis

☐ Infection ☐ Trauma ☐ Mental stress

☐ Puberty ☐ Pregnancy ☐ Menopause

☐ Drugs

- Preceding sore throat ☐ Yes ☐ No
- Alcohol intake ☐ Yes ☐ No
- Systemic illness ☐ Diabetes ☐ Hypertension ☐ HIV
- Pregnancy ☐ Yes ☐ No
- Lactation ☐ Yes ☐ No
- Past history of ☐ Photosensitivity ☐ Cutaneous malignancies
- Family history of Psoriasis ☐ Mother ☐ Father
  ☐ Sibilings ☐ Others
- Number of Children ☐ Male ☐ Female

Drug Intake

☐ Lithium	☐ NSAIDS	☐ Natural remedies
☐ Steroid withdrawal	☐ Beta blockers	☐ imalarials
☐ Amiodarone	☐ Digoxin	☐ Trazadone
☐ Penicillin	☐ Terfenadine	

Examination

General

Systemic: ☐ CVS ☐ RS ☐ Abdomen ☐ CNS

Dermatological

Auspitz sign ☐ No ☐ Yes

Clinical type Palmoplantar

Nail changes ☐ None ☐ Pitting ☐ Onycolysis

☐ Subungual hyperkeratosis ☐ Ridges

Joint involvement ☐ Yes ☐ No

Focal sepsis ☐ ENT ☐ Dental ☐ Others

Surface area involved ☐

Investigations **Hb:** **TC:** **DC:** **ESR:**

Urine albumin: Sugar: Deposits:

Blood sugar: Urea: Creat:

Sr.Calcium: LFT: VDRL:

Biopsy ELISA

Follow up

Date	No of Wks	UPPER LIMB PALMS				LOWER LIMB SOLES				PASI SCORE
		E	I	D	A	E	I	D	A	

E – Erythema, I – Induration, D – Desquamation, A – Area,

PASI – Psoriasis Area and Severity Index

Erythema / Induration / Desquamation scoring

0 – Nil

1 – Mild

2 – Moderate

3 – Severe

4 – Very severe

Area Scoring for Palms & Soles

0 – Nil

1 – Less than 10%

$$\text{PASI SCORE} = 0.2\,(E_H + I_H + D_H)\,A_U + 0.4\,(E_H + I_H + D_H)\,A_L$$

MASTER CHART

S.NO	AGE	SEX	DU	EX	FH	NI	FS	GR	PASI-0	PASI-4	PASI-8	PASI-12	PASI-24	AE
1	44	M	1	N	N	P	Y	TAZ	3.40	2.40	1.40	0.80	0.00	N
2	42	F	1	N	N	R	N	TAZ	4.40	4.60	3.60	2.60	1.60	N
3	63	F	0.5	ST	N	N	Y	TAZ	2.60	2.60	2.80	1.40	0.80	N
4	62	M	0.58	N	N	N	N	TAZ	3.00	2.20	1.80	0.80	0.00	N
5	29	F	3	ST	N	R	N	TAZ	2.60	3.40	2.20	0.60	0.00	Y
6	10	M	2	N	N	N	N	TAZ	4.00	3.00	1.80	1.60	1.20	N
7	38	M	4	N	N	P	Y	TAZ	2.40	3.00	3.20	1.60	0.80	N
8	55	M	3	N	N	N	N	TAZ	3.60	3.80	2.40	1.20	0.80	N
9	68	M	10	N	N	P	N	TAZ	3.20	2.60	2.00	0.80	0.60	Y
10	35	M	4	N	N	N	N	TAZ	4.60	2.80	2.20	1.00	0.40	N
11	28	M	2	N	N	O	N	TAZ	2.00	1.00	0.60	0.00	0.00	N
12	58	F	1	M	N	P	N	TAZ	3.50	3.30	3.00	1.60	0.60	N
13	49	M	2	N	N	R	N	TAZ	4.00	3.00	2.80	1.00	0.00	N
14	65	F	0.5	N	N	N	N	TAZ	2.20	1.40	1.20	0.60	0.00	N
15	50	F	1	N	N	P,R	Y	TAZ	3.40	2.80	2.20	1.00	0.80	Y
16	32	F	1.5	N	N	N	N	TAZ	3.20	2.20	1.60	1.00	0.00	N
17	27	M	0.67	N	N	P	N	TAZ	3.20	2.60	1.60	0.60	0.00	N
18	62	F	1	ST	N	P,R	N	TAZ	3.80	3.20	2.60	1.40	0.60	N
19	41	F	2	ST	N	N	N	TAZ	4.60	4.00	2.80	2.00	0.60	N
20	10	M	2	N	Y	R	N	TAZ	2.80	2.60	2.00	1.80	1.40	N
	43.4		2.138					TAZ	3.33	2.83	2.19	1.17	0.51	
21	14	F	0.58	N	N	R	N	PLA	2.60	1.80	1.40	0.60	0.20	N
22	68	F	0.08	N	N	O	N	PLA	2.80	1.80	2.00	1.60	2.20	N
23	53	M	0.5	N	N	N	N	PLA	1.80	1.60	2.00	2.40	1.80	N
24	38	M	0.75	N	N	N	N	PLA	3.80	2.20	1.80	2.00	2.40	N
25	63	M	15	ST	Y	R	N	PLA	2.80	2.60	2.00	1.60	1.40	N
26	47	M	0.25	N	N	P	N	PLA	3.60	2.80	2.40	2.00	1.80	N
27	40	M	4	N	N	N	N	PLA	2.60	2.40	2.20	2.60	1.80	N
28	23	M	13	N	N	P	N	PLA	4.80	4.40	3.60	3.20	3.40	N
29	30	M	3	CO	N	N	N	PLA	4.00	3.60	2.80	2.00	1.80	N
30	13	F	2	N	N	R	Y	PLA	2.00	1.80	1.40	0.80	0.40	N
31	36	M	0.25	N	N	N	N	PLA	2.60	2.00	1.80	0.60	0.20	N
32	49	F	0.67	N	N	O,R	N	PLA	3.20	2.60	2.00	1.40	0.80	N
33	51	M	2	N	N	N	N	PLA	2.80	2.60	2.80	2.20	2.60	N
34	13	M	1.5	N	N	N	N	PLA	3.20	2.20	1.60	1.47	0.80	N
35	50	F	5	N	N	P	N	PLA	4.80	4.60	3.80	4.20	4.00	N
36	85	F	0.5	N	N	N	N	PLA	3.60	3.40	3.00	2.80	2.60	N
37	12	M	3	N	N	P	Y	PLA	2.60	2.20	1.60	0.80	0.80	N
38	32	M	0.5	N	N	N	N	PLA	2.60	2.00	2.20	1.80	0.80	N
39	58	F	2	N	N	R	N	PLA	4..8	3.60	3.20	2.40	2.00	N
40	60	F	1	N	N	R	N	PLA	3.60	3.20	2.60	2.00	1.60	N
	41.75		2.779					PLA	3.15	2.67	2.31	1.924	1.67	
41	18	F	10	N	N	P	N	PUV	4.80	4.00	3.40	2.60	1.80	N
42	48	F	0.83	N	N	P	N	PUV	2.80	2.00	1.60	1.20	0.80	N
43	31	F	3	N	N	N	Y	PUV	1.80	1.60	0.60	0.60	0.40	N
44	35	F	0.17	N	N	P	N	PUV	2.80	2.20	1.80	-	-	Y
45	60	F	0.5	N	N	N	Y	PUV	4.20	3.20	2.80	1.80	0.80	N
46	45	M	20	N	N	O	N	PUV	4.80	3.80	2.60	2.00	1.20	Y
47	37	F	3	N	N	R	N	PUV	3.20	2.80	2.20	1.40	0.80	N
48	38	M	2	N	N	R	N	PUV	4.80	4.00	-	-	-	-
49	60	F	0.25	ST	N	R	N	PUV	3.00	2.80	1.80	1.20	0.60	N
50	42	F	1	N	N	N	N	PUV	4.80	3.60	2.80	1.60	0.80	N

MASTER CHART

51	40	M	0.5	N	N	R	N	PUV	2.20	2.20	1.20	0.80	0.20	N
52	50	M	3	N	N	N	N	PUV	2.80	2.00	1.60	1.20	0.60	N
53	50	M	0.42	N	N	R	Y	PUV	2.60	2.40	-	-	-	N
54	34	F	3	CO	N	N	N	PUV	2.80	2.00	1.40	0.80	0.00	N
55	52	M	0.5	N	N	N	N	PUV	3.20	2.40	1.80	1.40	0.80	-
56	39	F	4	N	N	R	N	PUV	2.00	1.80	1.40	-	-	Y
57	40	M	3	ST	N	N	N	PUV	2.60	2.20	1.60	0.80	0.60	N
58	28	F	1.5	N	N	P,R	N	PUV	4.20	3.60	2.20	1.40	0.80	N
59	28	M	4	N	N	N	N	PUV	2.80	1.60	1.00	0.80	0.40	N
60	42	M	4.5	N	N	N	N	PUV	3.80	2.60	2.00	1.80	0.40	N
	40.85		3.259					PUV	3.30	2.64	1.878	1.338	0.688	
61	63	F	5	N	N	P	N	STE	3.40	3.00	3.20	1.20	1.00	N
62	45	F	2	ST	N	N	N	STE	2.20	1.60	1.80	0.80	0.80	N
63	30	M	3	N	N	R	N	STE	3.20	2.60	2.40	0.80	0.80	N
64	62	M	3	N	N	N	N	STE	3.40	2.40	2.60	1.00	0.80	N
65	50	F	0.83	N	N	R	N	STE	4.40	4.00	3.60	2.80	2.40	Y
66	42	F	2	N	N	N	N	STE	3.20	3.00	3.20	1.80	1.40	N
67	49	M	0.75	N	N	N	Y	STE	3.20	2.40	2.80	3.20	2.40	Y
68	68	F	1	N	N	P	N	STE	2.60	2.40	2.60	1.00	0.80	N
69	37	M	1	N	N	R	N	STE	2.80	3.00	3.20	2.20	1.80	N
70	65	F	2	N	N	P,R	N	STE	3.80	3.60	3.60	3.20	2.40	Y
71	25	F	6	N	N	N	N	STE	2.60	2.40	2.60	1.00	0.80	Y
72	62	F	5	N	N	R	N	STE	4.40	4.00	3.80	2.00	1.80	N
73	61	M	1	N	N	N	N	STE	3.80	3.40	3.00	2.00	1.80	N
74	50	M	3	N	N	P	N	STE	4.00	3.20	3.40	1.60	1.00	Y
75	45	F	0.5	CO	N	R	N	STE	2.20	2.00	2.20	0.80	1.20	N
76	48	M	0.5	N	N	N	N	STE	2.80	2.40	2.60	1.00	0.80	N
77	38	F	0.5	N	N	N	Y	STE	3.80	3.20	3.20	1.80	1.20	N
78	43	M	1	N	N	N	N	STE	2.60	2.20	2.40	1.20	1.00	N
79	47	M	20	N	N	R	N	STE	4.60	3.80	4.00	4.40	3.60	N
80	60	M	0.25	N	N	N	N	STE	2.00	1.80	2.20	1.20	1.20	N
	49.5		2.917					STE	3.25	2.82	2.92	1.75	1.45	
81	43	M	5	N	N	P	N	DIT	3.20	3.00	2.40	1.40	1.60	N
82	47	M	1.5	N	N	N	N	DIT	4.40	3.60	2.60	1.40	0.60	N
83	23	F	3	N	N	N	Y	DIT	3.60	3.00	1.80	2.20	1.60	N
84	13	F	1	N	Y	P	Y	DIT	2.40	3.00	3.20	1.60	0.80	N
85	30	F	0.67	N	N	N	N	DIT	4.60	3.80	2.40	1.40	0.80	N
86	36	M	0.17	N	N	N	Y	DIT	2.00	1.00	0.60	0.00	0.00	N
87	59	M	1	N	N	N	N	DIT	1.80	1.60	0.60	0.60	0.60	N
88	36	F	2	ST	N	O,R	N	DIT	1.80	1.60	0.60	0.40	0.00	N
89	46	M	2	N	N	N	Y	DIT	2.60	3.40	2.80	2.00	0.60	N
90	18	M	1	N	N	N	Y	DIT	2.40	1.40	1.20	1.60	1.80	N
91	65	F	0.58	N	N	R	N	DIT	4.00	3.00	1.80	1.60	1.20	N
92	67	F	3	N	N	N	N	DIT	2.60	3.40	2.20	0.60	0.40	Y
93	34	F	3	N	N	R	N	DIT	4.40	4.60	3.80	2.40	1.60	Y
94	21	M	0.42	N	N	N	N	DIT	2.00	1.00	1.60	1.40	0.00	N
95	30	F	2	N	N	P	N	DIT	4.00	3.00	1.80	1.60	1.20	N
96	28	M	3	N	N	P	N	DIT	2.20	1.60	1.40	1.20	1.20	N
97	52	M	6	N	N	P	N	DIT	4.40	3.80	3.60	2.60	2.20	N
98	65	M	4	N	N	N	N	DIT	2.60	2.50	2.00	1.60	1.40	Y
99	60	F	7	M	N	P	Y	DIT	4.80	4.60	3.40	3.00	2.40	N
100	52	F	2	N	N	O,R	N	DIT	2.60	2.20	2.80	3.00	2.80	N
	41.25		2.417					TAZ	3.12	2.76	2.13	1.58	1.14	

KEY TO MASTER CHART

S.No – Serial Number
AGE – Age
SEX – Sex
- M – Male
- F – Female

DU – Duration of Psoriasis
EX – Exacerbating factors
- ST – Stress
- CO – Cold
- ME – Menopause
- N – Nil

FH – Family History
- Y – Yes
- N – NO

NI – Nail Involvement
- N – Nil
- PT – Pitting
- R – Ridging
- O – Onycholysis

FS – Focal Sepsis
- Y – Yes
- N – NO

GR – Group
- DIT –Dithranol
- PLA – Placebo
- STE – Steroid
- TAZ – Tazarotene
- PUV - PUVA

PA – PASI
- 0 – 0 week
- 4 – 4^{th} week
- 8 – 8^{th} week
- 12 – 12^{th} week
- 24 – 24^{th} week

AE – Adverse Effects
- Y – Yes
- N – NO